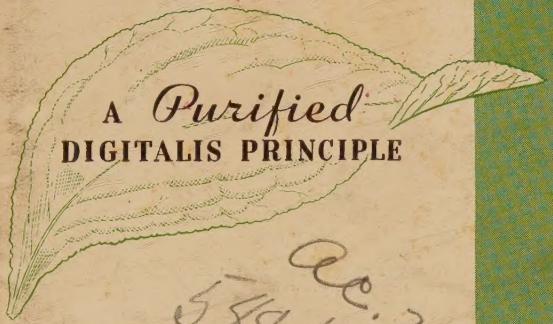
CRITERIA FOR THE CLASSIFICATION & DIAGNOSIS OF HEART DISEASE

WALTER G. KING
MED COL OF VA
RICHMOND, VA.



A *Purified*
DIGITALIS PRINCIPLE

QC.2
5884

VERODIGEN

Trade Mark

(GITALIN AMORPHOUS)

RARE CHEMICALS, INC.
NEPERA PARK, N. Y.
Manufacturing Chemists

CRITERIA FOR THE CLASSIFICATION & DIAGNOSIS OF HEART DISEASE

CRITERIA FOR THE
CLASSIFICATION & DIAGNOSIS OF HEART DISEASE

BY A COMMITTEE

JOSEPH H. BAINTON, M.D. W. C. MUNLY, M.D., M.C., U.S.A.
ROBERT L. LEVY, M.D. HAROLD E. B. PARDEE, M.D.
Chairman

OF THE HEART COMMITTEE OF THE NEW YORK TUBERCULOSIS AND HEALTH ASSOCIATION, INC.

ARRANGED IN CONFORMITY WITH THE NOMENCLATURE FOR CARDIAC DIAGNOSIS APPROVED BY THE AMERICAN HEART ASSOCIATION

SECOND EDITION

NEW YORK TUBERCULOSIS AND HEALTH ASSOCIATION

NEW YORK ✦ MCMXXIX

COPYRIGHT, 1929
BY NEW YORK TUBERCULOSIS AND HEALTH ASSOCIATION
All Rights Reserved

Published June, 1929

Printed in the United States of America
by J. J. Little & Ives Co.
New York City

PREFACE

FOR the most effective study of any kind of disease and in order that people working in different places may know that they are using the same terms for the same conditions, it is necessary to establish precise methods of labelling. This necessitates two things: (1) uniformity of nomenclature and (2) uniform criteria for using the nomenclature. Such labels, or nomenclature, should as far as possible be descriptive and descriptive only of the conditions which they indicate. The criteria should be precise definitions of the states named in the nomenclature.

To meet this demand with respect to heart disease the Committee on Cardiac Clinics of the Association for the Prevention and Relief of Heart Disease, the predecessor of the Heart Committee of the New York Tuberculosis and Health Association, prepared in 1923 a nomenclature covering diseases of the heart and blood vessels, which it adopted and introduced into its various clinics. With certain changes

this nomenclature was later adopted by the American Heart Association and has been used with success in all parts of the country.

Many different individuals using a nomenclature in different localities must have the same idea as to what a given label indicates if a uniform nomenclature is to achieve its purpose. This is all the more essential if statistical studies are to be made. The Heart Committee of the New York Tuberculosis and Health Association through its Research Committee is particularly concerned with such studies. It became obvious that after the completion of the nomenclature the next need was the establishment of definite criteria for diagnosis. To meet this need the Heart Committee appointed a committee to prepare Criteria for the Classification and Diagnosis of Heart Disease. This book, the result of their careful consideration over a period of nearly two years, has been approved and adopted by the Heart Committee.

While an effort has been made above to explain the *raison d'être* for this book, a very important service which we hope it

will also serve will be as a manual for physicians, to be used by them as an aid in diagnosis. Prepared as it has been by experts in heart disease these criteria are an extremely practical guide for correct diagnosis, and since proper management and treatment depend on correct diagnosis, its usefulness to the physician specially interested in heart disease should be great.

The Heart Committee realizes that thoughtful use of the criteria will undoubtedly suggest modifications, and hopes that those who use this book will not hesitate to send criticisms and suggestions for its improvement.

JOHN WYCKOFF, M.D.
Chairman, Heart Committee,
N. Y. Tuberculosis and Health Association

NEW YORK CITY

CONTENTS

	Page
Foreword from the Committee	xi
Introduction	1
Criteria for Nomenclature for Cardiac Diagnosis	3
Etiological	4
Anatomical	5
Physiological	8
Functional Capacity	11
Possible Heart Disease	11
Potential Heart Disease	11
Criteria for Diagnosis	13
Etiological	13
Anatomical	31
Physiological	63
Functional Capacity	87
Possible Heart Disease	92
Potential Heart Disease	92
Index	93

FOREWORD FROM THE COMMITTEE

THIS presentation of Criteria for the Diagnosis of Heart Disease is a development of ideas originally assembled in 1923 by the Committee on Cardiac Clinics of the Association for the Prevention and Relief of Heart Disease.[1] It was felt by the present Committee when approaching this subject again that it would be advisable to consider the whole field of cardiac diagnosis. In using the nomenclature of cardiac diagnosis as adopted by the American Heart Association a serious difficulty was encountered in arriving at a definition of the diagnósis itself. A given diagnostic term may mean a different thing to each of several physicians. For this reason each section begins with a brief definition of the

[1] Requirements for an ideal Cardiac Clinic and a System of Nomenclature of Heart Disease, by the Committee on Cardiac Clinics of the New York Association for the Prevention and Relief of Heart Disease. *Boston Medical and Surgical Journal*, 1923, CLXXXIX 762. This Committee consisted of Dr. E. T. Maynard, Dr. H. E B. Pardee, Dr. M. Rothschild, Dr. J. Wyckoff, and Dr. W. P. St. Lawrence, Chairman.

diagnosis under consideration and these definitions should be kept clearly in mind when reading the criteria for arriving at the diagnosis.

The criteria here described represent the opinion of a majority of the Committee. The section on Etiology was originally written by Captain Munly, that on Pathology by Dr. Levy, that on Physiology by Dr. Bainton and that on Functional Capacity by myself, but any statement upon which opinions differed was freely discussed until the majority opinion seemed evident.

Only what were considered the most important diagnostic criteria have been recorded. There are features other than those set down which may aid in reaching the diagnosis, but it is the opinion of the Committee that those which are here included are sufficient to form the basis of a diagnosis which will have a reasonable degree of accuracy. It was felt that further details would not appreciably increase the diagnostic accuracy and might lead to confusion.

Particular attention is called to the

suggestion of a uniform set of terms for describing heart sounds and murmurs. Much confusion results from the large vocabulary in use at present and it is hoped that these suggestions may pave the way to a simplification of the terminology of cardiac auscultation.

Much to the regret of this Committee Captain Munly was called for duty to San Francisco, and so was forced to resign. His place has been taken by Dr. Arthur C. DeGraff, whose work began with this revision, March, 1929.

HAROLD E. B. PARDEE
Chairman

INTRODUCTION

ONE of the aims of the Heart Committee of the New York Tuberculosis and Health Association is to study the natural history of the various types of heart disease. To accomplish this, data must be collected from many sources. Studies made in single clinics or groups are not necessarily representative of the whole. Comparable data can be obtained from different groups only when the same nomenclature of disease is used and the same criteria for diagnosis are applied.

We have accordingly adopted a standard nomenclature for cardiac diagnosis with slight modification,[1] and suggest the following diagnostic criteria as an aid in understanding and using this nomenclature.

[1] A nomenclature for cardiac diagnosis, *Am. Heart J.*, 2: 202, 1926. The terms "Myocarditis, acute" and "Myocarditis, chronic" have been condensed under the number 11 as "Myocarditis, (a) Acute; (b) Chronic."

The term "Fibrosis of the Myocardium" is given the number 12. Number 14 has been changed to "Myocardial Degeneration" with the subheadings, "(a) Fatty; (b) Parenchymatous."

14a. Hypotension has been added after 14. Hypertension.

It is realized that the criteria mentioned here may not be the only ones for arriving at the diagnosis, but it is believed that the use of these criteria will lead to accurate conclusions in the great majority of cases.

Attention also is called to the record forms which have been devised by the Heart Committee to aid in recording the facts on which the diagnosis must be based.[1]

[1] Clinical charts recommended by the Heart Committee of the New York Tuberculosis and Health Association. The plans for their use. *Am. Heart J.*, 2: 655, 1927.

A NOMENCLATURE FOR CARDIAC DIAGNOSIS [1]

FROM a study of the following tables of nomenclature it will be seen that a complete diagnosis is intended to include not only a statement of the structural changes in the heart but also one concerning the cause of such changes, one indicating the type of disturbance of physiological function, and finally one defining the functional capacity.

Such a comprehensive diagnosis obviously requires careful consideration of every aspect of each case and is therefore essential to sound prognosis and effective treatment.

Each diagnosis, to be complete, should include one or more titles from each of the groups, A, B, C, and D. Groups E and F, of course, require only reference to the etiological factor.

[1] Slightly modified from a nomenclature for cardiac diagnosis, *Am. Heart J.*, 2: 202, 1926.

A. Etiological

1. Unknown
2. Rheumatic fever [1]
   a. Polyarthritis
   b. Chorea
   c. Growing pains
   d. Tonsillitis
   e. Pharyngitis
   f. Others, as purpura, erythema nodosum, etc.
3. Syphilis [1]
4. Bacterial infection [1]
   (*Specify bacterium if possible.*)
   Here should be classified subacute infective endocarditis (Streptococcus viridans), chronic endocarditis, etc.
5. Thyroid [1]
   a. Hyperthyroidism
   b. Hypothyroidism
6. Toxic
   a. Bacterial toxin
   b. Mineral
   c. Vegetable
   (*Specify if possible.*)

[1] In the case of each of these conditions, it should be noted, if possible, whether the etiological factor is still active or is inactive.

7. Neurosis (Cardiac)
8. General systemic disease
   (*Specify the disease, e.g.*, arteriosclerosis, chronic nephritis, diabetes, emphysema, hypertension, anemia, etc.)
9. Trauma
10. Congenital developmental defect

B. Anatomical

1. Undiagnosed
2. Atrophy of heart
3. Enlargement of heart
4. Hypertrophy of heart
5. Dilatation of heart
6. Ventricular preponderance
   a. Right
   b. Left
7. Auricular hypertrophy
8. Cardiac thrombosis
9. Cardiac infarction
10. Rupture of heart
11. Myocarditis
    a. Acute
    b. Chronic

12. Fibrosis of myocardium
13. Myocardial infiltration
    a. Fatty
    b. Parenchymatous
14. Fatty degeneration
15. Endocarditis
    a. Acute
    b. Chronic
        (Include the continuous activity of a valvulitis, or papillary muscle, chorda tendineae or mural infection. Usually 15 would precede the 16 diagnosis.)
16. Cardiac valvular disease
        (Should be thought of as active or inactive as expressed under etiology.)
    a. Aortic insufficiency
    b. Aortic stenosis
    c. Mitral insufficiency
    d. Mitral stenosis
    e. Pulmonic insufficiency
    f. Pulmonic stenosis
    g. Tricuspid insufficiency
    h. Tricuspid stenosis

17. Congenital abnormality
    (*Specify lesion if possible.*)
18. Pericarditis, acute
    a. Serous
    b. Serofibrinous
    c. Purulent
19. Hydropericardium
20. Hemopericardium
21. Pneumopericardium
22. Adherent pericardium
23. Aortitis
    (Aortitis due to syphilis, etc., in contradistinction to 28, which includes arteriosclerotic changes in the aorta.)
    a. Without dilatation
    b. With dilatation
24. Tumor of
    (*Specify part affected.*)
25. Aneurysm of
    (*Specify affected vessel.*)
26. Embolism of
    (*Specify affected vessel.*)
27. Thrombosis of
    (*Specify affected vessel.*)

28. Arteriosclerosis of
(*Specify locality, as coronary arteries, cerebral arteries, etc.*)
29. Arteritis of (except aorta)
(*Specify locality.*)
30. Periarteritis of
(*Specify locality.*)
31. No circulatory disease
32. No cardiac disease

c. Physiological

1. Regular sinus rhythm
2. Ectopic rhythms (without tachycardia)
(This is to be differentiated from ectopic rhythms with tachycardia of the paroxysmal type. In the ectopic rhythms without tachycardia, it is usually believed that the new rhythm has developed because of a depression of the normal pacemaker. *Example:* Under this heading should be classified such a rhythm as nodal rhythm.)

3. Vagal arrhythmia
   a. Sinus arrhythmia
   b. Sino-auricular standstill
   c. Simple bradycardia
   d. Ventricular escape
   e. Wandering pacemaker
4. Sinus tachycardia (not paroxysmal)
5. Premature contractions
   a. Auricular
   b. Junctional
   c. Ventricular
   d. Unknown
6. Paroxysmal tachycardia
   a. Auricular
   b. Junctional
   c. Ventricular
   d. Unknown
7. Auricular flutter
   a. Paroxysmal
   b. Chronic
8. Auricular fibrillation
   a. Paroxysmal
   b. Chronic
9. Ventricular fibrillation

10. Auriculoventricular heart-block
    a. Partial block
        (1) Prolonged conduction time
        (2) Occasional dropped beats (irregular)
        (3) High grade block (2–1, 3–1, etc.)
    b. Complete block

11. Intraventricular block
    a. Partial
    b. Bundle-branch, right or left

12. Pulsus alternans

13. Valvular incompetency
    a. Mitral incompetency
    b. Tricuspid incompetency
    c. Pulmonic incompetency
    d. Aortic incompetency

14. Hypertension

14a. Hypotension

15. Congestive heart failure

16. Effort syndrome

17. Anginal syndrome

D. Functional Capacity

1. Patients with organic heart disease, able to carry on ordinary physical activity without discomfort.
2. Patients with organic heart disease, unable to carry on ordinary physical activity without discomfort.
   a. Activity slightly limited
   b. Activity greatly limited
3. Patients with organic heart disease and with symptoms or signs of heart failure when at rest, unable to carry on any physical activity without discomfort.

E. Possible Heart Disease

Patients who show abnormal signs or symptoms referable to the heart but in whom the diagnosis of heart disease is uncertain.

F. Potential Heart Disease

Patients without circulatory disease whom it is advisable to follow because of the presence or history of an etiological factor which might cause heart disease.

Examples

Heart Disease: A. Active syphilis; B. Aortitis with dilatation, aneurysm of the ascending aorta; C. Regular sinus rhythm; D. Class 2a; or

Heart Disease: A. Inactive rheumatic fever (chorea); B. Valvular disease, mitral insufficiency and mitral stenosis; C. Auricular fibrillation; D. Class 2b.

CRITERIA FOR DIAGNOSIS

A. Etiological

A CARDIAC diagnosis is incomplete without the etiology. Knowledge of the etiology helps to determine prognosis and treatment and often aids in the details of the diagnosis, particularly the anatomical diagnosis. Especially does the problem of prevention of heart disease center on etiology.

The history of past and present illness, the age of the patient, the structural lesions and, to a lesser extent, the functional disturbances are made use of in arriving at the etiological diagnosis.

The most common causes of heart disease are rheumatic fever, arteriosclerosis and syphilis. There are other less common causes but in many cases the etiology will be unknown or undetermined.

1. Unknown. The etiology should be stated as unknown in those cases which present either definite structural changes in the heart or evidence of abnormal

cardiac function for which no definite etiology can be determined.

A certain number of the cases of unknown etiology are probably due to rheumatic fever, the heart disease being its only manifestation. Such cases, in the absence of a definite rheumatic history, present the lesions typical of rheumatic heart disease and are of the age at which this is commonly found. Similarly, a certain number may be due to syphilis, though the syphilitic infection cannot be proven clinically.

Criteria for Diagnosis of Unknown Etiology.

The absence of a definite etiology in the presence of

(1) Structural lesion of the heart

(2) Abnormal cardiac function.

2. Rheumatic Fever. Rheumatic fever is probably a general infection which causes polyarthritis when it invades the joints, chorea when it attacks the brain, the rheumatic nodule when it lodges in the subcutaneous tissue, and rheumatic heart disease when it involves the heart. Rheumatic heart disease should be regarded as

one of the manifestations of rheumatic fever and not as a complication. Other evidences of rheumatic infection may be joint pains, muscle or growing pains, torticollis, tonsillitis, purpura and various erythemata. In some cases there may be persisting fever and other signs of infection without distinct clinical evidence of activity in any specific tissue. These rheumatic manifestations may occur singly or in various combinations at the same time, and are of varying duration.

Rheumatic fever usually shows itself in youth as muscle or joint pains, chorea, subcutaneous nodules or polyarthritis. The heart is often involved simultaneously or just following these manifestations but evidences of this involvement may be lacking for months or years. The heart is, in other cases, the first and apparently the only structure involved, so that rheumatic heart disease may occur without any other manifestation of rheumatic fever.

Rheumatic heart disease may be active or inactive. In its active stages it manifests itself as carditis, that is to say, acute inflammation of the pericardium, the

myocardium or the endocardium, alone or in combination. Pancarditis, or inflammation of the three structures of the heart, occurs frequently, though not always. Acute pericarditis may occur in the active stage and in healing leave adherent pericardium. Myocardial involvement may show itself in the active stages by evidences of acute myocarditis and after healing by permanent enlargement of the heart. Endocardial involvement shows itself in the active stages of rheumatic heart disease as acute endocarditis and valvulitis. Later, with healing, chronic structural changes in the valves remain, most commonly mitral insufficiency and stenosis, less commonly, aortic insufficiency and stenosis. Other lesions are more rarely found. There is often a latent period of varying duration from the onset of the original rheumatic infection to the discovery of cardiac valvular disease.

Rheumatic heart disease is generally a chronic disease with an extremely variable course. Some patients die after a short severe course; some show a prolonged course with continuously active heart

infection from the first attack until death; some show alternate periods of activity and quiescence and some, after one or more periods of active heart involvement, remain apparently free from infection for years.

The great majority of cases of rheumatic heart disease occur before the age of fifty and probably one half before the age of thirty.

The characteristic structural lesions are:

(1) Carditis.

(2) Cardiac valvular disease, most commonly mitral insufficiency and stenosis, less commonly aortic insufficiency and other valvular lesions.

(3) Adherent pericardium.

Criteria for Diagnosis of Rheumatic Heart Disease.

(1) History of polyarthritis, chorea, muscle or joint pains, or subcutaneous nodules, and evidence of a characteristic structural lesion of the heart.

(2) Evidence of a characteristic structural lesion plus a history of periods of recurrent fever and congestive heart failure.

(3) Evidence of carditis of the type associated with active rheumatic heart disease even in the absence of other rheumatic manifestations.[1]

3. Syphilis. The activity of the spirochetæ in the walls of the ascending aorta and occasionally in the heart muscle produces a characteristic type of heart disease.

The heart muscle is more commonly affected as a sequel of syphilitic disease of the coronary arteries than by direct infection. The ascending aorta and often the aortic valve are affected producing aortitis, aortic insufficiency and at times aneurysm. The other valves are seldom if ever involved. Involvement of the mitral valve is so rare that even if a patient presents mitral stenosis and a positive Wassermann reaction, the mitral stenosis

[1] Tonsillitis, purpura, and erythema may be rheumatic manifestations but need not be so. Therefore, unless they are associated with polyarthritis, chorea, muscle or joint pains, or subcutaneous nodules, the etiology should be written as "Unknown (tonsillitis)" or "Unknown (erythema nodosum)."

Patients with a history of rheumatic infection who present a systolic murmur at the apex without enlargement of the heart should be diagnosed as "Possible Heart Disease, E" and "Potential Heart Disease, F," rather than as "Rheumatic, Cardiac Valvular Disease, Mitral Insufficiency." (See page 92.)

should not be attributed to syphilis without post mortem proof.

The average time elapsing from the date of the original infection until the discovery of symptomatic heart disease is from fifteen to twenty years. Little is known of the course of the disease during this period. From the discovery of heart disease to death is usually a much shorter time. Syphilitic heart disease is rare before the age of forty and after the age of sixty.

The characteristic structural lesions are:

(1) Aortitis, particularly affecting the ascending portion. There may be dilatation of the arch.

(2) Aortic insufficiency.

(3) Aneurysm of the aorta.

Criteria for Diagnosis of Syphilitic Heart Disease.

(1) History of syphilitic infection and evidence of a characteristic structural lesion of the heart or aorta.

(2) A characteristic structural lesion of the heart or aorta, without a history of syphilis but with a positive Wassermann reaction.

(3) A characteristic structural lesion of

the heart or aorta together with evidences of syphilitic disease elsewhere, such as cerebrospinal syphilis, even in the absence of a positive Wassermann reaction or a history of syphilitic infection.

4. Bacterial Infection. This diagnosis is to include bacterial endocarditis due to known organisms, i.e., acute, subacute or chronic bacterial or infective endocarditis. The organisms likely to cause bacterial endocarditis are the Streptococcus viridans, the Streptococcus hemolyticus, the Staphylococcus aureus or albus, the Influenza bacillus, the Pneumococcus, the Gonococcus and the Meningococcus.

The course of bacterial endocarditis is acute, subacute or chronic, the duration, for purposes of classification, being arbitrary. The acute form is always fatal and takes the form of a vegetative or ulcerative endocarditis.

Signs of general sepsis entirely outweigh signs of cardiac involvement.

The subacute or chronic form of bacterial endocarditis is caused, in the majority of cases, by the implantation and growth of the Streptococcus viridans on previously

diseased valves or on a congenital defect. Its characteristic features are pallor, enlargement of the spleen, clubbing of the fingers, petechial hemorrhages, Osler's nodes, fever, fatigue and hematuria. Embolic phenomena are common. The blood culture may be positive or negative. Congestive heart failure usually occurs in the later stages.

Subacute bacterial endocarditis should be suspected in patients with heart disease who present pallor, clubbing of the fingers, enlargement of the spleen or embolic manifestations.

Criteria for Diagnosis of Bacterial Endocarditis.

Evidence of cardiac valvular disease or of a congenital abnormality associated with

(1) Signs of sepsis
(2) Positive blood culture
(3) Embolic phenomena.

5. Thyroid. a. *Hyperthyroidism.* All cases of hyperthyroidism show certain cardiovascular phenomena which are not due to heart involvement. The most important of these is sinus tachycardia

and forceful action of the heart. Only a certain proportion of patients with hyperthyroidism show signs of true heart disease.

The characteristic features of heart disease due to hyperthyroidism are enlargement of the heart, paroxysmal or permanent auricular fibrillation, and congestive heart failure.

Criteria for Diagnosis of Hyperthyroid Heart Disease.

(1) Evidence of hyperthyroidism and enlargement of the heart.

(2) Evidence of hyperthyroidism associated with abnormal cardiac function such as paroxysmal or permanent auricular fibrillation, or congestive heart failure.

b. *Hypothyroidism.* There is some evidence which indicates that myxedema may be associated with premature arteriosclerotic changes in the cardiovascular system. This may produce arteriosclerotic heart disease. While hypothyroidism may be attended by sluggish cardiac action and at times by congestive heart failure, further data are necessary to establish a

direct etiological relation between hypothyroidism and heart disease. Unless relief of heart symptoms can be clearly shown to be the result of medication with thyroid extract, hypothyroidism should not be considered the etiological factor.

6. Toxic. This etiologic diagnosis is to include those cases with abnormal cardiac function or structural cardiac disease clearly due to bacterial, mineral and vegetable toxins. These toxins should be specified. A striking example of a bacterial toxin is that of diphtheria; other local and general infections may have less marked effects. Phosphorus is a typical mineral poison and the toxic agent of uremia should probably be grouped here also. Tobacco, coffee and digitalis are typical vegetable toxins.

In the terminal stages of chronic diseases, a simple non-infectious endocarditis, which is considered to have a toxic origin, may be found. This is an anatomical diagnosis only. The etiological diagnosis "toxic" should not be made unless the toxin is clearly recognized. If not clearly estab-

lished, the etiology should be entered as "Unknown."

Criteria for Diagnosis of Toxic Heart Disease.

Evidence of a definite toxin (bacterial, mineral, vegetable) associated with

(1) Structural lesion of the heart

(2) Abnormal cardiac function.

7. Neurosis (Cardiac). Many patients who have no evidence of structural cardiac disease or definite toxic etiology, present one or more of the following symptoms or signs: breathlessness on exertion, fatigue, exhaustion, precordial pain, palpitation, giddiness, faintness, tachycardia, sinus arrhythmia, diffuse cardiac impulse, sluggish peripheral circulation, accidental murmurs and unstable blood pressure. Definite manifestations of a neurosis are to be found in the patient's history or examination.

This group of symptoms and signs has been variously termed "Effort Syndrome," "Neurocirculatory Asthenia" and "Irritable Heart." This etiology is probably not concerned in the production of true heart disease but is mentioned because it

produces symptoms like those found in true heart disease or may occur coincidentally with heart disease.

The characteristic features are that the symptoms or signs appear after less effort than would ordinarily produce them in healthy persons; that no cause can be found in the heart for these phenomena, and that other evidences of constitutional neuropathic stigmata appear.

Criteria for Diagnosis of Cardiac Neurosis.

Presence of symptoms and signs mentioned above in the absence of signs of structural cardiac disease.

8. General Systemic Disease. a. *Arteriosclerosis.* There is a type of heart disease commonly associated with general arteriosclerosis and due to its localization in the heart. The structural change is a fibrosis of the myocardium, probably resulting from inadequate blood supply to the cardiac tissues. At times, arteriosclerosis may be more advanced in the coronary than in other arteries, so that general arteriosclerosis may not be detected.

The aorta, coronary arteries and myocardium show sclerosis but the valves, though they may be deformed to some extent, are not often sufficiently affected to produce cardiac valvular disease.

Congestive heart failure or the anginal syndrome may be present. Abnormal rhythms, especially auricular fibrillation and flutter, often appear. Evidences of defective auriculoventricular or intraventricular conduction may be present. The arterial blood pressure need not be raised nor the heart enlarged.

For the purpose of this classification, the etiological diagnosis "Arteriosclerosis" should be given to those cases of heart disease which show definite evidences of general arteriosclerosis, even when such disorders as arterial hypertension, chronic nephritis, gout or diabetes are also present. Thus, given a case presenting evidence of general arteriosclerosis, enlargement of the heart and hypertension, the etiological diagnosis would be "Arteriosclerosis"; the anatomical diagnosis, "Enlargement of the heart," and the physiological diagnosis, "Hypertension." If chronic nephritis, gout

or diabetes are present, they are to be considered as "Other diseases," independent of the cardiac diagnosis.

Arteriosclerotic heart disease occurs chiefly in the sixth, seventh and eighth decades, being comparatively rare before forty-five years of age.

Criteria for Diagnosis of Arteriosclerotic Heart Disease.

General arteriosclerosis associated with:

(1) Sclerosis of coronary arteries.

(2) Evidence of fibrosis of myocardium either with or without the presence of congestive heart failure or the anginal syndrome.

(3) Occasionally coronary sclerosis is present without general arteriosclerosis.

b. *Hypertension.* It is frequently difficult or impossible to separate arteriosclerosis from hypertension as a cause of heart disease because they so often coexist.

Permanent hypertension may be associated with general arteriosclerosis or with chronic renal disease, or unassociated with any known disease (Essential hypertension).

Essential hypertension is a disorder in which the arterial blood pressure is permanently increased without known cause. It is regarded as a disturbance in function rather than in structure. In the early stages there may be no signs other than increased arterial blood pressure; later there may be enlargement of the heart. Where other cardiac signs and symptoms appear it is probable that arteriosclerosis of the aorta or coronary arteries has developed.

For the purposes of this classification, hypertension as an etiological diagnosis should be restricted to cases without demonstrable arteriosclerosis. When the latter is present, the etiological diagnosis should be entered as "arteriosclerosis," the hypertension being then a physiological diagnosis.

Criteria for Diagnosis of Heart Disease Due to Hypertension.

Persistent hypertension associated with enlargement of the heart.

c. *Other Systemic Diseases.* Heart disease may be associated with certain diseases

such as chronic nephritis, diabetes and gout. In such cases, arteriosclerosis or hypertension or both may be present and if so one of these should be the etiological diagnosis.

Long standing anemia is sometimes associated with cardiac enlargement.

There is insufficient evidence at present that measles, mumps, pertussis or chickenpox cause heart disease, although there is some evidence that scarlet fever and influenza do. More data are needed on the relation of heart disease to these and other infections.

Any acute infection may aggravate the existing cardiac disease and at times may actually cause it, but unless the evidence is conclusive the etiological diagnosis should be entered as "Unknown" and the acute infection entered under a heading "Other diseases."

The causal relation of foci of chronic infection to heart disease is not proven. The etiological diagnosis should be entered as "Unknown" unless bacteriological proof is available.

In all cases of heart disease in which any uncertainty exists as to cause, the etiologi-

cal diagnosis should be stated as "Unknown" and the questionable etiological factor entered under a heading "Other diseases."

9. Trauma. Traumatic heart lesions occur as the result of penetrating wounds, great strains, blows or severe crushing injuries.

10. Congenital Developmental Defect. See Criteria for Anatomical Diagnosis, paragraph 17.

B. Anatomical

In a majority of cases of heart disease, it is possible to make a satisfactory anatomical diagnosis. The various diagnostic methods employed are: (1) complete physical examination, including the determination of temperature and blood-pressure; (2) electrocardiographic examination, as an aid in appraising the nature and extent of structural changes in the myocardium; (3) roentgen-ray examination, using teleroentgenography, fluoroscopy, or both, to ascertain the position, size and shape of the heart and great vessels; (4) Wassermann test, (5) blood count and (6) other tests, when indicated. The findings are correlated with the facts concerning etiology and with functional disturbances.

To compile a complete catalogue of the signs of the numerous varieties of heart disease is beyond the scope of these notes. An attempt has been made to set down those features regarded as of fundamental diagnostic importance, together with a few of such secondary criteria as may, on

occasion, prove helpful. Those signs which are considered most useful for diagnosis are marked with an asterisk (*). The anatomical classification followed is, with very slight changes,[1] the one approved by the American Heart Association.

The following general suggestions are made:

In making the examination it is best to follow a routine, including inspection, palpation, percussion and auscultation. Electrocardiographic and roentgen-ray examination and other special tests are made as indicated.

Inspection should include a search for precordial bulging, point of maximum impulse and other localized impulses or points of retraction, for the presence of edema, cyanosis, pallor, petechiae, subcutaneous nodules and clubbing of the fingers, and examination of the neck veins for unusual engorgement or pulsation.

Palpation should first be used to locate the apex impulse. The point furthest downward and outward at which can be felt a distinct thrust forward serves to indicate

[1] Cf. footnote, page 3.

the position of the left border of the heart. Particularly when the heart is enlarged, this point furnishes a more useful guide to the left border than does percussion. The point of maximum impulse usually lies mesial to this point. Abnormal shocks and thrills, the presence of edema and enlargement of the liver and spleen are also made out by palpation. The pulse rate and character and the consistency of the arterial wall should be determined.

Percussion serves to outline the area of cardiac dullness and that of the basal vessels.

Auscultation enables the examiner to ascribe character to the heart sounds and to detect and describe murmurs. Heart sounds and murmurs are described by adjectives denoting intensity, quality and duration. The description of murmurs includes, in addition, the time of occurrence in the cardiac cycle and the site of maximum intensity. On occasion it is useful to note the direction of their transmission and the effect of respiration and posture upon them. Changes in the qualities of sounds and murmurs observed on succes-

sive examinations should be noted. Certain special procedures for eliciting murmurs will be referred to under the proper captions. A diagnosis of cardiac arrhythmia should be attempted by auscultation.

Different observers often use different terms to describe the same sounds and murmurs. This is due in part to the variable ability of individuals to classify auditory impressions, but in large measure to the multiplicity of terms employed. Many of these are synonymous and it is believed that an attempt to standardize descriptive terminology may prove helpful.

The following list of descriptive terms, all in common use, will serve to characterize most heart sounds and murmurs. If in exceptional conditions the terms here given seem inadequate, it is suggested that the most nearly satisfactory ones from this list be used, together with such others as may be necessary. In the table the terms in larger type (FAINT) are recommended for routine use. Those in smaller type (Weak) are regarded as synonyms for those in capitals immediately preceding them and may be used as

alternates if it is thought that the recommended term is inadequate.

TABLE OF TERMS TO BE USED IN DESCRIBING HEART SOUNDS AND MURMURS

HEART SOUNDS

Intensity	Quality	Duration	Time
NORMAL	NORMAL	NORMAL	
FAINT	SHARP	SHORT	
Weak	Snapping		
Distant	Valvular		
Muffled			
LOUD	BOOMING	PROLONGED	
Accentuated	Muscular		
Increased			
ABSENT	SPLIT		
Replaced by a Murmur			
	REDUPLICATED		
	RINGING		
	Clanging		

MURMURS

FAINT	BLOWING	SHORT	SYSTOLIC
Soft	Soft		
MODERATE	HARSH	MODERATE	EARLY SYSTOLIC
	Rough		
	Coarse		
LOUD	MUSICAL	LONG	LATE SYSTOLIC
	RUMBLING		DIASTOLIC
	CRESCENDO		EARLY DIASTOLIC
	DECRESCENDO		MID-DIASTOLIC
			PRESYSTOLIC (Late Diastolic)

Cardiorespiratory murmurs are sometimes confusing in that they may simulate those indicating valvular disease. They are

usually heard at the apex or over the body of the heart, at the lung margins. They are almost always systolic in time and vary in intensity during the phases of respiration. Holding the breath during a forced expiration sometimes diminishes or abolishes them. Their loudness may be increased by exercise.

Unexplained murmurs have been called "functional," "accidental" or "hemic." They are almost always systolic in time, are often inconstant and vary in intensity with change in posture. The most common is a harsh systolic murmur at the pulmonic area, which is apt to be best heard with the patient lying down and also during expiration. A blowing apical systolic murmur is also frequently heard. Such murmurs do not indicate cardiac disease and are of no clinical significance. The mode of their production is unknown.

1. Undiagnosed. It is rarely necessary to file under this heading.

2. Atrophy of the Heart. This condition is associated with wasting diseases

or anemia. The diagnosis is seldom made except at necropsy.

3. Enlargement of the Heart. This term is used to include combined hypertrophy and dilatation. These conditions rarely occur separately and the relative importance of each in a given case must be decided chiefly by clinical observation.

Signs:

*Apex beat to the left of its normal position. The left border of a normal heart does not usually lie more than 1 cm. to the left of the mid-clavicular line.[1]

*Increased area of cardiac dullness.

*Increased size of the heart by roentgen-ray examination. In a teleroentgenogram, the total transverse diameter of the normal heart does not usually exceed one-half of the internal transverse diameter of the chest.

Precordial bulging.

4. Hypertrophy of the Heart. Hypertrophy is characterized by enlargement

[1] This is the same distance from the mid-line as is the mid-point of the clavicle.

of the heart and a strongly heaving apex impulse. Slight degrees of hypertrophy cannot, at times, be recognized.

5. Dilatation of the Heart. This is characterized by enlargement of the heart and evidence of congestive heart failure. It is usually accompanied by murmurs denoting relaxation of the mitral or tricuspid orifices, or both.

6. Ventricular Preponderance. This refers to an abnormal relation between the mass of right and left ventricular muscle. It is inferred from the direction of the waves of the Q R S group of the electrocardiogram, after correlation with the position and size of the heart. The larger the heart, the more importance may be ascribed to the electrocardiographic changes.

(a) Right. The main deflection of the Q R S group in Lead I is downward, and R is larger in Lead III than in Lead II. A vertical position of the heart exaggerates this relation of the electrocardiographic waves.

(b) Left. The main deflection of the Q R S group in Lead III is downward and R

is larger in Lead I than in Lead II. A transverse position of the heart exaggerates and may even be the sole cause of this relation of the electrocardiographic waves.

7. Auricular Hypertrophy. This is combined with dilatation and is seen most frequently in mitral stenosis, the left auricle being the one enlarged.

Signs:

*Prominence of the left auricle on roentgen-ray examination.

*P waves of the electrocardiogram are broad, high and notched.

8. Cardiac Thrombosis. This refers to thrombosis within the auricular or ventricular cavities. It is a diagnosis by inference, occurring in the presence of auricular fibrillation or marked congestive heart failure or after cardiac infarction. Emboli may break off from the cardiac thrombus and lodge either in the lungs or in the peripheral circulation. Under these circumstances the probability of the diagnosis is strengthened.

9. Cardiac Infarction. Infarction of the myocardium occurs almost invariably following thrombotic closure of a coronary artery. Its presence should be suspected after coronary occlusion has occurred. For the diagnostic criteria, see Thrombosis of Coronary Artery, page 58.

10. Rupture of the Heart. This diagnosis is rarely made except at necropsy.

11. Myocarditis. a. *Acute.* Evidence of acute infection in the heart muscle is found most frequently in rheumatic fever, occasionally in other infectious diseases and in the various forms of general sepsis due to pyogenic bacteria. Syphilitic and tuberculous lesions occur rarely. The diagnosis is made largely by inference, in the presence of an appropriate etiology.

Signs:

*Tachycardia;

*Changes in the form of the electrocardiogram indicative of myocardial damage [see Fibrosis of myocardium, page 41].

Fever.

Leucocytosis, except in tuberculous or syphilitic cases.

Congestive heart failure of gradual onset and progressive course.

Cardiac arrhythmia, particularly auricular fibrillation, flutter or heart block.

Enlargement of the heart.

Faintness of the first heart sound.

b. *Chronic.* The chronic is distinguished from the acute phase chiefly on the basis of duration. Chronic myocarditis implies continued activity of the infection over a period of time, the duration, for purposes of classification, being arbitrary. In chronic myocarditis there is usually some degree of cardiac enlargement.

12. Fibrosis of the Myocardium. This term is employed to denote the condition in which the heart muscle shows fibrotic changes, following acute or chronic inflammation, or associated with arteriosclerotic or degenerative processes. Congestive heart failure is usually, though not always, present in some degree. The diagnosis can be made only in the absence of an acute inflammatory or toxic etiology.

Signs:

*Modification of the first heart sound (it may be faint, short, prolonged, split or reduplicated).

*Changes in the form of the electrocardiogram, indicative of myocardial damage. Such changes are (1) inversion of the T wave in Lead I, Leads I and II, Leads II and III, or in all leads; (2) notching or slurring of Q R S; (3) widening of Q R S beyond the normal upper limit of 0.10 second. These findings, singly or in combination, may or may not be present. If found, they afford evidence of definite heart muscle impairment, but do not indicate whether this is due to fibrosis or to some other process.

Cardiac arrhythmia, particularly permanent auricular fibrillation or heart block.

Enlargement of the heart may or may not be present.

Gallop rhythm.

13. Fatty Infiltration. A condition sometimes observed in obese individuals. Cardiac enlargement is often present. Examination of the heart at necropsy

shows an increase of fatty tissue between the muscle bundles.

14. Myocardial Degeneration. This refers to a degeneration of the muscle fibers due to certain bacterial or chemical toxins. The diagnosis is usually made at necropsy, but may be suspected clinically in the presence of appropriate etiological factors.

Myocardial degeneration may be
(a) Fatty
(b) Parenchymatous.

Signs:
Tachycardia.
Cardiac arrhythmia.
Faintness of the first heart sound.
Systolic murmur at the apex, usually faint.

15. Endocarditis. a. *Acute.* Evidences of acute inflammation of the endocardium are found most frequently in rheumatic fever, less often in the various forms of bacterial sepsis. The diagnosis is made largely by inference, often in the absence of characteristic physical signs, but must

be made only in the presence of an appropriate etiologic factor.

Signs:

*Murmurs of cardiac valvular disease. These are especially significant when their first appearance is noted while the patient is under observation. The changing character of murmurs, particularly in the direction of increasing intensity, may indicate active endocarditis.

*Fever.

Embolic phenomena (petechiae are frequent in the bacterial forms).

b. *Chronic.* The chronic is distinguished from the acute phase chiefly on the basis of duration. Chronic endocarditis implies continued activity of the infection over a period of time, the duration, for purposes of classification, being arbitrary. Subacute or chronic bacterial endocarditis is to be included under this diagnostic heading.

16. Cardiac Valvular Disease. Endocarditis may terminate by healing without residue or may result in permanent

scarring with valvular deformity. Valvular defects are distinguished by characteristic signs.

a. *Aortic Insufficiency.* This diagnosis can be made only in the presence of a characteristic murmur.

Signs:

*Diastolic murmur, blowing or musical in quality, and of a pitch higher than that of the usual first heart sound. It may be very faint, often better heard with the ear directly applied to the chest than with the stethoscope. It is loudest (1) in the third or fourth interspaces at the left border of the sternum, or (2) over the sternum itself at this level, or (3) in the second or third interspace at the right border of the sternum. The murmur is best heard with a relatively slow heart rate, with the breath held in expiration and with the patient erect or bent forward.

Enlargement of the heart, especially to the left.

Collapsing (Corrigan) pulse.

Large pulse pressure, with lowered diastolic pressure.

b. *Aortic Stenosis.* Unless the murmur of aortic insufficiency is also present, the diagnosis of aortic stenosis must be made with great caution.

Signs:

*Loud, harsh, systolic murmur at the aortic area, transmitted upward. Such a murmur at the aortic orifice may occur in the absence of actual stenosis, e.g., due to aortitis or sclerosis of aorta, with or without dilatation of the aorta.

*Systolic thrill, best felt in the second interspace to the right of the sternum.

Faint or absent aortic second sound.

Anacrotic pulse, with slow rise and broad summit.

c. *Mitral Insufficiency.* Not all apical systolic murmurs denote mitral valvular disease. Sometimes the murmur is evidence of incompetency, due to dilatation of the valve orifice. Sometimes it is of the accidental or unexplained variety. With a history of rheumatic infection and especially when associated with other valvular lesions or with cardiac enlargement the

murmur is more likely to indicate valvular disease.

Signs:

*Systolic murmur, constantly present, best heard at or just outside the region of the cardiac apex, either following or replacing the first heart sound and usually transmitted toward the axilla. Its quality may be blowing, harsh or musical, with relatively high pitch.

*Enlargement of the heart. This may be of varying degree but without it the presence of valvular disease is uncertain.

d. *Mitral Stenosis.* A positive diagnosis can be made only in the presence of a characteristic murmur.

Signs:

*The characteristic murmur is a low-pitched, diastolic rumble, often with presystolic, crescendo accentuation. Sometimes this latter is the only murmur heard. The murmur may be only mid-diastolic; in the presence of auricular fibrillation it is often heard only during the first part of diastole. Often the murmur

of mitral insufficiency is also present. The murmur of mitral stenosis can sometimes be made out only with the patient lying on his back or left side. It is almost always louder in these positions than when erect. Acceleration of the heart rate by slight exercise, such as changing from the recumbent to the sitting posture ten times, will frequently make plain a murmur which is otherwise inaudible.

*Sharp or snapping first sound at the apex. In the presence of a sharp apical first sound and the murmur of mitral insufficiency, early mitral narrowing may be suspected.

*Left auricular enlargement, observed by means of the roentgen ray.

In certain cases of aortic insufficiency, a diastolic murmur (Austin Flint murmur) is heard at the apex, in the absence of mitral valve disease. This murmur cannot be differentiated by its qualities from that due to organic mitral narrowing. In a given case, if aortic insufficiency is of rheumatic etiology, an apical diastolic murmur is more likely to be due to mitral stenosis. If, on the other hand, the aortic insufficiency is syphilitic, it is fair to

assume that the mitral valve is anatomically intact and that it is a Flint murmur which is heard. If there is a sharp first heart sound, the diagnosis of mitral stenosis is more probable.

e. *Pulmonic Insufficiency.*
f. *Pulmonic Stenosis.*

These are rare conditions, except as congenital lesions. They will be discussed under that heading.

g. *Tricuspid Insufficiency.* This is more frequently due to valvular incompetency than to valvular disease. As a valvular lesion, it is usually a part of rheumatic heart disease and is associated with other valve lesions.

Signs:

*Prolonged systolic murmur of blowing quality, sometimes harsh, best heard at the level of the fifth chondrosternal junction, either over the sternum or just to the right of it.

*Cardiac enlargement to right and to left.

*Presence of other murmurs of cardiac valvular disease.

*Systolic, expansile pulsation of an enlarged liver.

*Systolic pulsation of the external jugular vein.

h. *Tricuspid Stenosis.* A rare condition of rheumatic origin. The murmur is similar to that of mitral stenosis but is best heard in the fourth right intercostal space near the sternal border.

17. Congenital Abnormality. In the diagnosis of congenital heart disease, two distinct problems are present. A congenital must be distinguished from an acquired lesion, and a precise anatomical differentiation of the various defects may be attempted. The recognition of the presence of a congenital lesion is usually relatively easy. The numerous combinations of defects and the variability of the physical signs make hazardous a precise definition of the structural anomalies. In the majority of cases, only a probable anatomical diagnosis can be made.

Cyanosis and clubbed fingers, if present, are helpful diagnostic features, but their frequency has been overemphasized. Other

developmental defects may be present elsewhere in the body.

Murmurs are present in practically every case. They are usually systolic in time and harsh in quality, and may or may not be accompanied by a thrill. Though often audible over a wide area, the points of maximum intensity are either at the base or over the body of the heart. Accidental or unexplained murmurs often occur in these situations. A systolic murmur maximum at the base, if due to congenital abnormality, is most frequently due to pulmonic stenosis, less commonly to aortic stenosis. A systolic murmur heard loudest over the body of the heart, especially along the left sternal border, is often indicative of a patent interventricular septum. In view of the fact that pulmonic stenosis and patency of one of the septa are so often associated, it is not a simple matter to decide upon which lesion the murmur depends. Cardiac enlargement, especially to the right, is present in most cases of patent septum.

A patent ductus arteriosus is recognized by the presence of a long, loud continuous

murmur with systolic, or systolic and diastolic intensification, best heard in the second or third left interspace and in the back. Pulmonic stenosis is usually also present.

Dextrocardia may be recognized by physical examination. Confirmation is afforded by roentgen-ray examination and a characteristic electrocardiogram in which P, Q R S and T waves are all inverted in Lead I. The usual waves of Lead II are seen in Lead III, and vice versa.

Signs of Congenital Heart Disease:

*Murmurs as described above.

*Abnormal cardiac outline on roentgen-ray examination.

*Cyanosis.

Clubbed fingers.

Changes in the form of the electrocardiogram. Right ventricular preponderance and Q R S group of unusually high voltage are frequently found.

18. Acute Pericarditis.

a. *Serous.*

Signs:

*Increased area of cardiac dullness, more easily noticeable at the right border. With large effusions it often extends to the left of the apex beat. With small amounts of fluid, only the basal dullness may be increased.

*Pericardial friction rub. This may often be heard in the presence of effusion.

*Faint heart sounds.

*Enlarged cardiac area by roentgen-ray examination, often with characteristic pear shape. Cardiac enlargement, however, may give a picture closely resembling pericardial effusion.

b. *Serofibrinous.*

Signs:

*Pericardial friction rub, superficial and scratchy, usually heard at the base or over the body of the heart. The friction sound frequently has a to-and-fro character, but may be only systolic or only diastolic.

c. *Purulent.* The signs are as in Serous Pericarditis (page 52), with the addition of more definite signs of sepsis.

19. Hydropericardium. The physical signs are as given for serous pericarditis, except that no pericardial friction rub is heard. It is seen in the presence of advanced congestive heart failure. The fluid has the characteristics of a transudate.

20. Hemopericardium is usually a result of trauma or of rupture of the heart or aorta. The physical signs are as given for serous pericarditis (Paragraph 18a).

21. Pneumopericardium. This is most often a result of trauma, and is usually complicated by serous, purulent or bloody effusion. Tympany replaces the cardiac dullness and, if fluid is present, splashing or churning sounds may be heard. On roentgen-ray examination the air within the pericardium is apparent.

22. Adherent Pericardium. The diagnosis is often overlooked. The signs vary according to whether the adhesions are between the two pericardial layers or between the parietal pericardium and the mediastinum, pleura or diaphragm.

Signs:

*Cardiac enlargement, especially if greater than might be anticipated from the valvular lesions present.

*Fixation of the apex beat. On turning the patient from the left to right side, no shifting of the apex beat is observed.

*Fixation of the right border of cardiac dullness. On turning the patient from side to side, the right border does not move more than 1 cm.

*Fixation of the electrical axis of the electrocardiogram in spite of alterations in posture.

Systolic retraction in the region of the apex with diffuse cardiac impulse.

Diastolic shock over body of heart.

Diaphragm phenomenon (Broadbent). This is a systolic retraction in the eleventh interspace posteriorly on the left side. The sign may occur in thin-chested individuals with great cardiac hypertrophy without adhesions between pericardium and diaphragm.

23. Aortitis is to be distinguished from sclerosis of the aorta. Its etiology is an

infection, usually syphilis. In syphilitic aortitis, a positive Wassermann test may be expected in only about 80 per cent of cases.

a. *Without Dilatation.* At this stage, symptoms are more helpful than signs, e.g., the anginal syndrome or paroxysmal dyspnea. In a syphilitic patient, the diagnosis may sometimes be suspected from the presence of a harsh, systolic murmur at the aortic area.

b. *With Dilatation.*

Signs:

*Increase in aortic shadow is seen in roentgen-ray films and exaggerated aortic pulsation under the fluoroscope. In syphilis it is the ascending aorta which is most frequently involved.

*Harsh, systolic murmur, loudest at the aortic area.

*Ringing quality to the aortic second sound. Often the intensity is increased. This is in contrast to aortic stenosis in which the aortic second sound is faint.

Increased basal dullness.

Sometimes a systolic thrill may be felt at the aortic area, and also in the carotid artery.

24. Tumor of the Heart. This is a rare condition. The diagnosis should be suspected if cardiac signs or symptoms develop in the course of malignant disease elsewhere in the body, especially signs of hemorrhagic pericardial effusion or obstruction of a large vessel.

25. Aneurysm of the Thoracic Aorta. Aneurysm of the aorta may be a latent condition without either symptoms or signs. The roentgen-ray examination, particularly with the fluoroscope, serves to establish the diagnosis.

Signs:

*Roentgen-ray examination may reveal diffuse dilatation or sacculation, involving the ascending, transverse or descending arch. The descending aorta is less frequently involved.

*Localized pulsation over the upper chest.

*Signs of bronchial compression.

*Abnormal dullness at base of heart.

Signs of aortitis and aortic insufficiency.

Tracheal tug.

Inequality of the brachial pulses.

Inequality of the blood pressure in the two arms.

Evidence of recurrent laryngeal nerve paralysis.

25a. Aneurysm of Other Arteries. The diagnosis is made by the finding of a pulsating tumor but the details scarcely come within the limits of this work.

26. Embolism of a Coronary Artery. A rare occurrence, not clinically distinguishable from thrombosis of coronary artery.

26a. Embolism of other Arteries. This is diagnosed by various signs, depending upon the situation of the artery. The details of each condition are without the limits of this work.

27. Thrombosis of a Branch of a Coronary Artery. The diagnosis is made by both symptoms and signs. These vary according to the size and location of the occluded vessel.

Symptoms and Signs:

*Precordial or substernal pains, sudden in onset and agonizing in character, with variable radiation (see Anginal Syndrome, page 84). Administration of nitrites usually affords no relief. Pain is not invariably present. Without pain this attack produces the appearance of shock and is usually associated with pulmonary edema.

*Sharp fall in blood pressure.

*Faintness of the heart sounds, often with gallop rhythm.

*Pericardial friction rub. This does not occur until several hours after the attack. It may be transient or may not be heard at all.

*Fever appearing twelve to twenty-four hours after onset and rarely high.

*Leucocytosis. This may often be found within the first twelve hours. If other causes for leucocytosis can be excluded, its presence affords presumptive evidence of myocardial infarction.

*Changes in the form of the electrocardiogram, involving T wave, Q R S group or both. If followed from day to day the form of the curves may alter with

change in the state of the affected area of myocardium.

*Cardiac arrhythmia may make its appearance. Ventricular premature beats, ventricular tachycardia and auricular fibrillation or flutter are the most common disturbances observed.

Congestive heart failure may appear, particularly associated with edema of the lungs.

27a. Thrombosis of other Arteries. This is diagnosed by various signs depending upon the situation of the artery. The details are without the limits of this work.

28. Sclerosis of the Aorta. The diagnosis is made by the presence of the same signs described for Aortitis (23), the finding of arteriosclerosis elsewhere, and the absence of syphilitic etiology.

28a. Sclerosis of the Coronary Arteries. Occasionally no diagnostic signs are present. It is often though not invariably associated with general arterioscle-

rosis. The condition may manifest itself in several ways.

Signs:

Fibrosis of myocardium.

Anginal syndrome.

Thrombosis of a branch of a coronary artery.

Cardiac arrhythmia, paroxysmal or permanent.

28b. Sclerosis of other Arteries. This is diagnosed by various signs depending upon the situation of the artery. If palpable, the artery is felt to be thickened, tortuous or "beaded." The other details are without the limits of this work.

29. Arteritis. An acute inflammatory reaction involving the arterial coats, often leading to occlusion of the lumen. It has a patchy distribution and is a rare condition.

30. Periarteritis Nodosa. A rare condition, not often diagnosed clinically.

C. Physiological

1. Regular Sinus Rhythm. This is the normal rhythm of the heart originating in the sinus node. The average rate in healthy individuals lies between 60 and 80 per minute at rest. The rate tends to become slower when lying down and more rapid on standing and after exercise.

2. Ectopic Rhythms (without tachycardia). The rhythm of the heart is maintained by the initiation of the impulses from centers other than the sinus node.

a. *Nodal Rhythm.* Depression of the sinus node may result in the auriculoventricular node assuming control of the rhythmic beat of the heart. The ventricular rate is relatively slow.

The diagnosis is made only by the electrocardiogram. The P wave is abnormal in form. The P-R interval is reduced or the P wave may be submerged in or follow the Q R S group. In some records all three of these changes in the P-R interval may be observed.

b. *Idioventricular Rhythm.* When impulses from the auriculoventricular node fail to reach the auriculoventricular bundle, the latter or some other site in the ventricles becomes the pacemaker for the ventricles.

Arrhythmias. In the clinical study of arrhythmias auscultation of the heart is a more accurate way of obtaining knowledge of the irregularity than palpation of the pulse. In the majority of cases, clinical examination will lead to a correct diagnosis. Graphic records, especially the electrocardiogram, will sometimes be necessary to obtain exact information.

3. Vagal Arrhythmias. Changes in vagal tone may influence the activity of the sinus node causing variations in the rate and rhythm of the heart.

a. *Sinus Arrhythmia.* Clinically this should be suspected when the heart slows progressively for a short series of beats and the slowing is followed by a gradual quickening. This variation is usually continuous. Ordinarily, quickening occurs with

inspiration, slowing with expiration. At times the phases of sinus arrhythmia do not coincide with the phases of respiration. Occasionally the irregularity may be very marked.

b. *Sino-auricular Standstill.* Standstill of the whole heart for a single beat, both auricles and ventricles, may occur because the sinus node is quiescent or the impulse from it is blocked before reaching the auricles. Clinically it should be suspected on noticing a dropped beat by auscultation, but the true character can be recognized only by graphic methods. It must be distinguished from the pause associated with an early premature contraction or from heart block with dropped beats.

c. *Simple Bradycardia.* This is a slow sinus rhythm with a rate less than 60 per minute which tends to become slower when lying down and more rapid on standing and after exercise.

d. *Ventricular Escape.* An independent beat of the ventricles may occur when the impulse initiated at the sinus node is

delayed or is prevented from reaching the ventricles. The independent beat usually originates in the auriculoventricular node or bundle. The diagnosis is made only by graphic methods. The ventricular complexes of the electrocardiogram are supraventricular in type (as contrasted with the complexes due to ventricular premature contractions). They are not preceded at the usual interval by a P wave.

e. *Wandering Pacemaker.* This is recognized only in the electrocardiogram where the form of the P wave and the auriculoventricular conduction time vary from beat to beat.

4. Sinus Tachycardia. This is a rapid regular sinus rhythm with a rate of 100 per minute or more, tending to become slower when lying down and more rapid on standing and after exercise.

5. Premature Contractions. The heart's action is interrupted by a contraction originating prematurely in a focus outside of the sinus node. The interruptions

may occur regularly or irregularly. When a premature contraction interrupts a regular sinus rhythm, it is recognized on auscultation as a beat occurring before the expected time of the next normal beat. It is followed by a longer pause than normal. This pause ends in a beat which is usually unduly loud. These characteristics are somewhat modified when a premature contraction occurs during an abnormal rhythm or when it is completed before the next beat of the normal rhythm (interpolated premature beat) so that there is no long pause.

On palpation of the radial artery a premature beat or an intermission of pulse will be found to correspond with the premature heart sounds.

Exercise, by increasing the heart rate, will often abolish premature contractions, whereas the irregularity due to auricular fibrillation will persist.

The point of origin of premature contractions can often be determined with accuracy only by the electrocardiographic record, though polygraphic curves often suffice.

a. *Auricular.* The P wave is changed in contour and follows the preceding P at less than the usual interval of the record. The P-R interval may be shortened, normal or lengthened. The Q R S group, though changed in form, resembles that of the normal beats.

b. *Junctional (Auriculoventricular Nodal).* The P wave is usually abnormal in form. The P-R interval is shortened or the P wave may be submerged in or follow Q R S. The Q R S group, though changed in form, resembles that of the normal beats.

c. *Ventricular.* The ventricular complex is abnormal and premature. The P wave is not usually seen, being submerged in Q R S or in the T wave of the premature beat. Rarely it precedes the premature Q R S by a very short interval. The Q R S group is notched or slurred, its base increased in width and its voltage large. The T wave is usually large and always opposite in direction to the main deflection of Q R S in the lead showing the largest Q R S deflection.

d. *Unknown as to Site of Origin.*

6. Paroxysmal Tachycardia. This condition is a rapid and regular succession of ectopic beats which occur in paroxysms of varying duration. The rate may lie between 110 and 200 per minute. The beginning and ending of the attacks are sudden and are usually recognized by the patient.

Clinically, paroxysmal tachycardia may be suspected when there is a rapid, regular rhythm with a rate of over 150 per minute, the rate being unaffected by position, rest or exercise. Pressure upon either vagus nerve in the neck may terminate the attack, but otherwise will not slow the rate.

The paroxysms originate in some point outside the sinus node:

(a) *Auricle*
(b) *Junctional tissues*
(c) *Ventricle*
(d) *Unknown site of origin*

Differential diagnosis can be made only by the electrocardiogram, the point of origin being identified in a manner analogous to the method of identifying the origin of premature contractions.

7. Auricular Flutter. This condition is a disturbance of auricular activity due to a circus movement within the auricular muscle, continually following the same path at a constant rate. The auricles contract regularly at a rate which lies between 200 and 350 per minute. The ventricular rate is usually one-half, but may be one-third or one-fourth of the auricular rate, depending upon the facility of A-V conduction. The ventricular rhythm may be regular or irregular, or changing from one to the other.

Clinically, it should be suspected when the ventricular rate is about 150 per minute and the rhythm regular. The rate is usually unaffected by change in position, rest and moderate exercise. There is generally marked temporary slowing of the ventricles on vagal pressure in contrast to the absence of this effect in paroxysmal tachycardia. When vagal pressure causes slowing the rhythm may become decidedly irregular.

In the electrocardiogram, auricular activity is manifested by waves which differ from the normal P wave in being

broader and usually following each other in such rapid succession that no isoelectric period is demonstrable. These waves are usually best seen in Leads II and III, and are remarkably constant in rate, form and amplitude for the same patient. The ventricular complexes are of supraventricular type and distort the auricular waves.

Auricular flutter may be

(a) *Paroxysmal*

(b) *Permanent (chronic)*

8. Auricular Fibrillation. This condition is a disturbance of auricular activity due to a circus movement travelling at a varying rate and not following a constant path. The rate of this movement is between 350 and 500 per minute. The ventricles contract irregularly, the rate depending in large measure upon the facility of auriculoventricular conduction.

The ventricular rate without treatment generally lies between 90 and 160 per minute. Occasionally rates as low as 40 to 60 may be obtained, especially when the heart is under good digitalis control.

At times the irregularity of the rhythm is so slight that it may escape detection without very careful observation.

Clinically, it should be suspected when the heart beat is so grossly irregular that no evidence of a dominant rhythm can be obtained. Often there is a tendency to alternating periods of rapid and slow beats. Exercise increases the heart rate and accentuates the irregularity, in contrast to the usual result when arrhythmia is due to premature contractions.

In the electrocardiogram, the P wave is absent, auricular activity being represented by a series of wavelets which vary in form, amplitude and rate. The ventricular complexes with rare exceptions are spaced irregularly.

Auricular fibrillation may be

(a) *Paroxysmal*

(b) *Permanent* (*chronic*)

9. Ventricular Fibrillation. This is almost invariably a terminal event, a phenomenon of the dying heart. It is recognized only by the electrocardiogram which presents a series of large irregular

waves like distorted ventricular premature contractions.

10. Auriculoventricular Block. The conduction of the impulses passing from auricles to ventricles is impaired.

a. *Partial Block.*

(1) Prolonged conduction time can be detected only by a graphic record. The record shows the auriculoventricular conduction time beyond the normal limit of 0.20 seconds.

(2) Occasional dropped beats and (3) high grade block are variable degrees of the same process and often occur at different times in the same patient.

Clinically, dropped beats due to heart block may be suspected when auscultation reveals a slow rate, usually between 40 and 60, with a regular rhythm interrupted at times by a pause equal to double the normal.

A dropped beat due to partial heart block may be confused with the long pause which follows an early premature ventricular contraction. In the latter in-

stance, by careful auscultation at the apex, the sounds of the premature beat can be heard early in the period of intermission of the pulse, while in the former condition there is absolute silence throughout the pause. A graphic record will make the differential diagnosis.

Dropped beats due to sino-auricular standstill and those due to partial heart block can be differentiated only by graphic records.

The electrocardiogram of partial block usually shows the P-R interval increased and at times the P wave may fail to be followed by a ventricular complex. The ventricular beats may be absent only occasionally or with fair frequency. Sometimes they may be dropped regularly in a definite ratio to the auricular beats, occurring for example after every fourth, third or second auricular beat, which is commonly spoken of as 4-1, 3-1, 2-1, heart-block.

b. *Complete Block.* Clinically, this should be suspected when the ventricular rate is under 40 per minute and the rhythm is

regular. The ventricular rate varies but little or not at all with change of position or after exercise. At times there may be prolonged periods of ventricular standstill during which syncope and convulsions occur (Adams-Stokes syndrome).

The electrocardiogram shows regularly recurring ventricular complexes at a slow rate which have no relation to auricular activity. The latter may be slow or fast or may show fibrillation or flutter.

11. Intraventricular Block. In this condition conduction is defective below the bifurcation of the auriculoventricular bundle. Impaired conduction may manifest itself in either the right or the left bundle-branch ("bundle-branch block") or in any subdivision of the intraventricular conducting system ("partial intraventricular block"). It is usually only one feature of severe myocardial damage. Clinically, it may be suspected when the patient shows evidence of myocardial fibrosis.

The diagnosis can be made only by the electrocardiogram. It is not possible to distinguish between lesions of the intra-

ventricular conducting system and lesions lying within the ventricular walls.

Attempts have been made to associate various types of electrocardiographic changes with specific anatomical lesions. The terms partial bundle-branch block, arborization block and bundle-branch block have been applied in this connection. Correlation of electrocardiographic and pathological findings has not furnished a sound basis for these distinctions, so that it seems advisable to regard all these various electrocardiographic abnormalities merely as evidence of ventricular disease, i.e., an impaired ventricular muscle.

a. *Partial.* The Q R S group is increased in duration beyond 0.10 second and is notched or slurred. Its voltage may be large or small. The T waves may be upright or inverted in any lead or combination of leads.

b. *Bundle-branch Block.* The graphic changes commonly described as bundle-branch block are distinctive and the criteria are therefore given under a separate heading. It should be emphasized that

even this term is an electrocardiographic designation and does not always denote a specific anatomical lesion.

Right bundle-branch block produces an electrocardiogram in which the features of a levogram predominate: (1) The Q R S group is notched or slurred; (2) its duration is prolonged beyond 0.12 second; (3) the main deflection is directed upward in Lead I and downward in Lead III, and (4) the T wave is in the opposite direction to the main deflection of Q R S in the leads of large excursion.

Left bundle-branch block produces an electrocardiogram in which the features of a dextrogram predominate: (1) the Q R S complex is notched or slurred; (2) its duration is abnormally prolonged (beyond 0.12 second); (3) the main deflection is directed downward in Lead I and upward in Lead III, and (4) the T wave is in the opposite direction to the main deflection of Q R S in the leads of large excursion.

12. Pulsus Alternans. The rhythm is regular but every other beat is weak. This can be detected by palpation of the pulse.

By the auscultatory method of taking blood pressure the sounds appear to be evenly spaced, with regular alternation of loud and feeble sounds. Nearing the upper level of systolic pressure the feeble sounds disappear because the strong beats have a higher systolic pressure than the weak.

This condition may be suspected when weak and strong pulse beats alternate. It must be distinguished from the regular occurrence of premature contractions. Pulsus alternans can be demonstrated by a sphymographic tracing but not by an electrocardiographic record.

13. Valvular Incompetency. Presumably due to weakness or dilatation of the supporting ring, valvular incompetency is to be distinguished from chronic valvular disease with insufficiency in that there is no anatomic lesion of the valve itself.

a. *Mitral Incompetency.* Evidence of mitral incompetency is afforded by a systolic murmur which has the same characteristics as the murmur described for insufficiency of the valve. The differentia-

tion is made largely by inference, keeping in mind the following features:

(1) Mitral incompetency occurs commonly in congestive failure and especially if marked enlargement is present.

(2) In the absence of rheumatic etiology, when there is disease of the myocardium or aortic valves, the murmur is probably due to incompetency and not to valvular disease.

(3) When there is a rheumatic history with aortic valvular disease, it may be impossible to decide whether the murmur is produced by disease of the mitral valve or by functional incompetency. Without marked enlargement or congestive failure valvular disease is the more likely cause.

(4) In the presence of mitral stenosis the murmur should be considered as due to insufficiency.

At times the murmur must be differentiated from the accidental systolic murmurs heard at the apex (see pages 35, 36).

b. *Tricuspid Incompetency.* The signs are not distinguishable from those described for tricuspid insufficiency due to valvular disease. The diagnosis as in mitral

incompetency is made largely by inference. Tricuspid incompetency is secondary to preexisting mitral disease or incompetency, or to obstruction in the pulmonary circulation.

c. *Pulmonic Incompetency*. The murmur of pulmonic incompetency is indistinguishable from that due to pulmonic insufficiency. It may be heard in association with marked mitral stenosis and congestive heart failure. It is called the Graham Steell murmur.

d. *Aortic Incompetency*. A diastolic murmur like that of aortic insufficiency may occur in the absence of aortic valvular disease.

14. HYPERTENSION. This may be diagnosed when the systolic pressure is found to be above 140 mm. of mercury or the diastolic above 90, under conditions which exclude the pressure raising effects of muscular and nervous activity. These figures are for adults.[1] The figures for

[1] Report of Joint Committee on Mortality of the Association of Life Insurance Medical Directory and the Actuarial Society of America, N. Y., 1925.

children are not so high and vary with age.[1]

14a. Hypotension. This may be diagnosed when the systolic blood pressure is below 110 in men or below 100 in women. The Committee has selected these arbitrary figures in the absence of adequate data.

15. Congestive Heart Failure. This is a syndrome which is a result of the failure of the heart as a pump. Evidences of capillary stasis and impeded venous return are present and give rise to the following phenomena:

(a) *Abnormal Dyspnea.* In mild degrees of failure this is present only on exertion. In severe failure dyspnea may be present even at rest.

(b) *Orthopnea.* This usually is present in moderate or advanced failure.

(c) *Basal Râles.* The presence of basal râles is usually one of the early signs of

[1] Faber, H. K. and James, C. A. The range and distribution of blood pressure in normal children. *Am. J. Dis. Child.*, 22:7, 1921.

heart failure. As failure increases the râles become more numerous. Occasionally basal râles may be absent, even in extreme failure.

(d) *Diminished Vital Capacity.* In general this is more and more diminished as failure progresses.

(e) *Distended Neck Veins.* Impeded venous return to the heart results in distention of all the large veins. The prominent jugular veins are but part of a general venous distention. Distended neck veins are observed in moderate or advanced congestive failure.

(f) *Enlargement of the Liver.* The same causes which produce distention of the neck veins may also cause enlargement and tenderness of the liver. When congestive heart failure has been long continued some enlargement of the liver may remain after the failure has been relieved.

(g) *Edema.* In the lesser grades of congestive failure, edema may be present about the ankles, appearing at the end of the day and disappearing over night. In

general, as failure increases, the edema becomes more marked and more extensive. Occasionally, however, there may be but little edema with severe heart failure.

(h) *Hydrothorax*
(i) *Ascites*

Evidence of free fluid in the pleural or abdominal cavities is not found except in the more marked degrees of congestive failure.

In children with active rheumatic heart disease signs of marked congestive failure are often lacking. Instead there are apt to be evidences of a general infection with dyspnea, tachycardia, fever, low-grade leucocytosis, wasting, progressive anemia and weakness, together with signs of active cardiac involvement.

16. Effort Syndrome (see A. Etiological, 7, Neurosis, page 24). This is considered a disturbance of function without pathological basis in the heart. It is manifested by an exaggeration of the normal response to physical exertion, mental effort or emotional stress.

The signs and symptoms are out of all

proportion to the energy expended and are as follows: easy fatigation, faintness, tremulousness, palpitation, heart pain, abnormal dyspnea on exertion and tachycardia. The apex beat is often vigorous. Accidental murmurs may be present.

Effort syndrome may be found in an individual who has real heart disease. In such a case, it will be necessary to distinguish between the symptoms due to neurosis and those due to organic disease of the heart.

17. Anginal Syndrome. The cardinal symptom is substernal or precordial pain occurring in attacks. Under different circumstances the pain varies in severity from a slight discomfort to an intense, gripping, vise-like agony. Instead of pain, some patients feel substernal distress, constriction or a sense of suffocation.

This pain sometimes has a marked tendency to radiate. The most frequent radiation is down the left arm, even to the finger tips. It may radiate into the right shoulder and arm, up into the neck or occiput, to the teeth, or into the upper

abdomen. Occasionally the pain will start at a distal point and travel back to the precordium.

In many cases the pain is initiated by physical effort, excitement, eating or exposure to cold wind. At times no immediate cause for the onset of the attack can be discovered.

In severe attacks the appearance of the patient is noteworthy. Intense suffering may be evident or there may be an appearance like surgical shock. There is often a profuse perspiration. There are no characteristic physical signs.

The diagnosis of anginal syndrome is incomplete without the inclusion of some feature which may constitute a cause. Such features may occur singly or in combination. The following list includes the most important:

Structural Changes (Anatomical).

Sclerosis of aorta
Aortitis
Aneurysm
Sclerosis of coronary artery
Thrombosis of coronary artery
Pericarditis

Abnormal Function (Physiological).
Congestive heart failure, when acute or severe
Cardiac arrhythmia
Cardiac neurosis
Toxic (Etiological).
Tobacco
Coffee

In occasional cases no causal feature can be determined. The most common associated lesions are diseases of the aorta and coronary arteries.

D. Functional Capacity

At the present time there is no clinical test which will accurately measure the functional capacity of the heart. This section of the diagnosis refers, then, to the functional capacity of the cardiac patient, as modified by his cardiac disease. Only an approximate estimate of this functional capacity is possible, and the most useful guide is found in the patient's ability to perform physical activity. There are numerous factors which may make it difficult or even impossible to estimate the effect of cardiac failure upon the ability to exercise. Such factors are the presence of an acute disease, convalescence, physical weakness, anemia, arthritis and others.

The functional classification should not be influenced by the anatomical diagnosis or by the prognosis. It should depend solely on the functional capacity of the patient at the time of the examination. Furthermore, the management of the patient should not be determined by

the functional capacity alone but by its correlation with the etiology, the anatomical lesions and the physiological disturbances.

The functional classification depends upon the patient's ability to carry on ordinary physical activity in so far as this is modified by the functional capacity of the heart.

The term "ordinary physical activity" is meant to include *all of the activities which would be expected* of the patient had he a normal heart, giving due consideration to age, sex, weight and habits of exercise. Not only are the slighter, more common exertions important for rating ability, but especially the more severe exertions which the patient, except for the presence of heart disease, would be expected to perform without undue discomfort.

In estimating the patient's response to effort, a comparison is made between the expected normal and the present ability, as limited by the cardiac condition. Usually this estimate may be based entirely upon a careful history of the patient's symptoms on effort. Is distress

produced by walking on the level, or up a grade, or by running, or by climbing stairs, or by other types of effort? If the anginal syndrome or undue breathlessness appears after effort and extracardiac factors are not thought responsible, the functional capacity may be considered reduced. If the physician is in doubt as to the rating by this method, an observation of the patient after he has performed exercise may be helpful.[1]

In general, in the presence of congestive failure, the more marked the physical signs (p. 81) the greater is the reduction of functional capacity. At times, however, there is considerable variance between the degree and number of the physical signs and the amount of reduction in the functional capacity.

If active heart infection is present there is usually some degree of congestive failure. The functional capacity is, as a rule, reduced in proportion to the severity of the infection, but the presence of active

[1] The Committee is of the opinion that while exercise tests are of value as aids in estimating functional capacity, there are at present insufficient data on the use of these tests to warrant their inclusion in these notes.

heart infection should never be used as a guide to the functional classification. Frequently it will be impossible to determine the functional capacity of patients with active infection.

Classification of Patients

Class 1. Patients with organic heart disease able to carry on ordinary physical activity without discomfort. Ordinary physical activity does not cause undue fatigue, palpitation, dyspnea or chest pain. Patients in this class do not show physical signs of congestive heart failure and rarely signs of active heart infection.

Class 2. Patients with organic heart disease unable to carry on ordinary physical activity without discomfort.

a. *Activity Slightly Limited.* Ordinary physical activity causes undue fatigue, palpitation, dyspnea or chest pain. Patients in this class rarely show physical signs of congestive heart failure or signs of active heart infection.

b. *Activity Greatly Limited.* Less than ordinary physical activity causes fatigue,

palpitation, dyspnea or chest pain. Patients in this class usually show one or more physical signs of congestive heart failure, or the anginal syndrome or signs of active heart infection.

Class 3. Patients with organic heart disease and with symptoms or signs of heart failure at rest, unable to carry on any physical activity without discomfort. There is fatigue, palpitation, dyspnea or chest pain at rest. Patients in this class show marked physical signs of congestive heart failure or the anginal syndrome, or signs of active heart infection.

E. Possible Heart Disease [1]

Patients who show abnormal signs or symptoms referable to the heart but in whom the diagnosis of heart disease is uncertain should be diagnosed as "Possible Heart Disease, Class E."

F. Potential Heart Disease

Patients without heart disease, whom it is advisable to follow because of the presence or history of an etiological factor which might cause heart disease, should be diagnosed as "Potential Heart Disease, Class F." In such cases the etiological factor should be stated.

[1] A patient who has both abnormal signs and symptoms and a history of an etiological factor which might cause heart disease should be classified as Possible Heart Disease and Potential Heart Disease.

INDEX

A

Adherent pericardium, 7, *54*
Anatomical classification, *5*
Anatomical criteria, 31
Anemia, *5*, 29
Aneurysm, 7, *57*
Anginal syndrome, 10, 84
Aortic incompetency, 10, 80
Aortic insufficiency, 6, *45*
Aortic stenosis, 6, 46
Aortitis, 7, *55*
Arrhythmia, sinus, 9, 64
Arrhythmia, vagal, 9, 64
Arrhythmias, 64
Arteriosclerosis, *5*, 8, *25*
  of aorta, 60
  of coronary arteries, 60
  of other arteries, 61
Arteriosclerotic heart disease, 27
Arteritis, 8, 61
Atrophy of heart, *5*, 36
Auricular fibrillation, 9, 71
Auricular flutter, 9, 70
Auricular hypertrophy, *5*, 39

Auricular paroxysmal tachycardia, 9, 69
Auricular premature contractions, 9, 68
Auriculoventricular heart block, 10, 73

B

Bacterial endocarditis, 20
Bacterial infection, 4, 20
Bradycardia, simple, 9, 65
Bundle-branch block, 10, 76

C

Cardiac neurosis, 5, 24
Cardio-valvular disease, 6, 44
Chorea, 4, 14, 17
Classification of patients, 90
Congenital abnormality, 7, 50
Congenital developmental defect, 5, 30
Congenital heart disease, 50
Congestive heart failure, 10, 81

D

Diabetes, 5, 26, 29
Dilatation, 5, 38

E

Ectopic rhythm, 8, 63
Effort syndrome, 10, 83

Embolism, 7, 58
Emphysema, 5
Endocarditis acute, 4, 6, 43
Endocarditis bacterial, 4, 44
Endocarditis chronic, 6, 44
Enlargement of heart, 5, 37
Erythema nodosum, 4, 15, 18
Etiological classification, 4, 5
Etiological criteria, 13
Etiology, 13
Extra systoles (see premature contractions)

F

Fatty degeneration, 6, 43
Fatty infiltration, 6, 42
Fibrillation auricular, 9, 71
Fibrillation ventricular, 9, 72
Fibrosis of myocardium, 6, 41
Functional capacity, 11, 87

G

Growing pains, muscle or, 4, 15, 17

H

Heart block, auriculoventricular, 10, 73
Heart block, intraventricular, 10, 75
Heart failure, congestive, 10, 81

Hemopericardium, 7, 54
Hydropericardium, 7, 54
Hypertension, 5, 10, 27, 80
Hypotension, 10, 81
Hyperthyroid heart disease, 22
Hyperthyroidism, 4, 21
Hypertrophy, 5, 37
Hypertrophy auricular, 5, 39
Hypothyroidism, 4, 22

I

Idioventricular rhythm, 64
Infarction, cardiac, 5, 40
Infiltration, fatty, 6, 42
Insufficiency, aortic, 6, 45
Insufficiency, mitral, 6, 46
Insufficiency, pulmonic, 6, 49
Insufficiency, tricuspid, 6, 49
Intraventricular block, 10, 75

J

Junctional paroxysmal tachycardia, 9, 69
Junctional premature contractions, 9, 68

M

Mitral incompetency, 10, 78
Mitral insufficiency, 6, 46

Mitral stenosis, 6, 47
Myocarditis, acute, 5, 40
Myocarditis, chronic, 5, 41
Myocardium, fibrosis of, 6, 41

N

Nephritis, chronic, 5, 26, 29
Neurosis, 5, 24
Neurosis, cardiac, 24
Nodal rhythm, 8, 63
Nodule, rheumatic, 14

P

Paroxysmal tachycardia, 9, 69
Periarteritis, 8, 61
Pericarditis, acute, 7, 52
Pericardium, adherent, 7, 54
Pharyngitis, 4
Physiological classification, 8
Physiological criteria, 63
Pneumopericardium, 7, 54
Polyarthritis, 4, 14, 17
Possible heart disease, 11, 92
Potential heart disease, 11, 92
Premature contractions, 9, 66
Preponderance, ventricular, 5, 38
Pulmonic incompetency, 10, 80
Pulmonic insufficiency, 6, 49

Pulmonic stenosis, 6, 49, 51
Pulsus alternans, 10, 77
Purpura, 4, 15, 18

R

Regular sinus rhythm, 8, 63
Rheumatic fever, 4, 14
Rheumatic heart disease, 14
Rheumatic nodule, 14
Rhythm, ectopic, 8, 63
Rhythm, idioventricular, 64
Rhythm, nodal, 8, 63
Rhythm, regular sinus, 8, 63
Rupture of heart, 5, 40

S

Sclerosis 25-27, 60, 61
Simple bradycardia, 9, 65
Sino auricular standstill, 9, 65
Sinus arrhythmia, 9, 64
Sinus rhythm, regular, 8, 63
Sinus tachycardia, 9, 66
Standstill, 9, 65
Stenosis, aortic, 6, 46
Stenosis, mitral, 6, 47
Stenosis, pulmonic, 6, 49, 51
Stenosis, tricuspid, 6, 50
Syphilis, 4, 18

Syphilitic aortitis, 55
Syphilitic heart disease, 18

T

Tachycardia, paroxysmal, 9, 69
Tachycardia, sinus, 9, 66
Thrombosis, 5, 7, 39, 58
Thyroid, 4, 21
Tonsillitis, 4, 15, 18
Toxic etiology, 4, 23
Toxic heart disease, 24
Toxin bacterial, 4, 23
Toxin, mineral, 4, 23
Toxin, vegetable, 4, 23
Trauma, 5
Traumatic heart lesions, 30
Tricuspid incompetency, 10, 79
Tricuspid insufficiency, 6, 49
Tricuspid stenosis, 6, 50
Tumor of heart, 7, 57

U

Unknown etiology, 4, 13

V

Vagal arrhythmia, 9, 64
Valvular disease, 6, 44

Valvular incompetency, 10, 78
Ventricular escape, 9, 65
Ventricular paroxysmal tachycardia, 9, 69
Ventricular premature contraction, 9, 68
Ventricular preponderance, 5, 38
Vital capacity, 82

W

Wandering pacemaker, 9, 66

C0-AVA-316